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CHANGES IN THE ORAL CAVITY DUE TO BRUCELLOSIS

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For the last 10 years a great deal of attention has been focused on the problem of brucellosis. This disease was first reported in the Soviet Union almost simultaneously by Professors Kryukov and Smirnov, Docents Zdradovskiy and Ngegonrv, and then by Isaakyan, Makhviladze, and Kandelaki.

The main sources of human infection are sheep, goats, large-horned cattle and swine which have brucellosis.

Due to the polymorphism of the clinical course of the disease, it is very frequently not recognized and is diagnosed as malaria, military tuberculosis, polyarthritis rheumatica, vulgar sepsis, typhoid fever, croup, etc.

In pronounced cases where all symptoms characteristic for brucellosis are present, the disease can be clinically determined with accuracy. For cases which do not have the complete syndrome of brucellosis, we are forced to adopt different methods of investigation. Thus these cases are very important from the epidemiological standpoint.

In the evacuation hospital located at the Tropical Institute during the World War II there was a series of cases of brucellosis in which the symptoms appeared in various degrees. We were commissioned to determine whether changes in the oral cavities of these patients have diagnostic and prognostic value for brucellosis.

We studied 79 cases of brucellosis during which the patients were investigated from their hospitalization to their discharge.

During the examination of the oral cavity of these patients, we discovered a rather rare disease of the gums, namely, gingivitis, s. parodontitis marginalis atrophicans denudans.

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At the onset of the disease, the gums reddened very slightly and formed a swelling about midway between the interdental papillae on the buccal (labial) or the palatal (lingual) surface of the teeth. The disease was frequently overlooked at this stage since the changes occurred without pain. The marginal gingiva around the swelling was hyperemic dark red in color. Bleeding of the gums was frequently observed. Interdental papillae and other surfaces of the gums were not affected.

We did not notice any other deviations from the norm, either in the front or the oral cavity proper, except for the tongue, which in most cases was covered with a thick white coating.

The described changes of the gums were observed on both jaws in the region of the cuspids on the labial and lingual surfaces and in the region of the palatal roots of the upper molars.

We considered these changes are attributable to brucellosis -- the disease which changed the reactivity of the body. Gingivitis causes changes in the gums depending on the length of time elapsed since the onset.

We noted these changes in the oral cavity in 70 (88.5 percent) of the 79 cases. In the remaining 9 (fresh cases) there were no noticeable deviations from the norm.

Nearly all of the patients afflicted with this disease of the gums had an unusually fine subgingival tartar deposited irregularly and in limited quantities on either the buccal (labial) or lingual (palatal) surface of the teeth. This tartar was usually dark brown, and occasionally black. It was firmly attached to the cement of the teeth so that it was difficult to scrape off.

The gingivitis observed in cases of brucellosis, in contrast to ordinary atrophic gingivitis and pyorrhea alveolaris, was accompanied by the formation of pathological pockets, secretion of pus, disturbance of the statics of the teeth, and considerable deposits of hard dental tartar.

It must be assumed that the atrophic changes that we observed stemmed from other causes, that is, the neurotrophic factor.

Ya. S. Pekker considers the last as predominant in every atrophic change of the gingiva in the oral cavity. Ye. I. Tarakanov attributed to the neurotropic factor the greatest influence in the process of brucellosis.

For illustration we cite an extract from a case history.

Patient S, born in 1914, entered the evacuation hospital on 6 September 1943 with diagnosed brucellosis. At the end of 1942 he had lived in B., where he drank raw milk and ate clabber. He complained of general asthenia, headaches, excessive irritability, and muscular pains. His temperature was subfebrile. The disease struck suddenly 10 July with increase in temperature, headache, severe pain in the back, and profuse perspiration. The mucous membrane in both the front and the oral cavity proper was pale except in the region 7 6 1 6 7 where there was hyperemia of the marginal gingiva. The marginal gingiva was not scaling. The submaxillary lymph nodes were enlarged. Midway between the interdental papillae, in the region mentioned above, there was the characteristic swelling. There was also subgingival tartar but only on the palatal surface. There were no pyorrheal pockets.

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The tonsils were normal. There were no blocks. Baring of the roots was not appreciable and occurred only in the region 7 6 1 6 7. There was no displacement or loosening of the teeth nor were there any teeth which had not penetrated through the gums. The tongue was moist and covered with a white coating.

Autovaccine, local therapy, and salicylic preparations, daily washing with hydrogen peroxide and painting of 5 percent methylene blue on the afflicted parts were administered twice daily.

The patient remained in the evacuation hospital from 6 September 1943 to 11 January 1944. The first remission lasted from 9 September to 23 September 1943. The patient's state of health was again satisfactory. It is interesting to note that by 20 September, despite the fact that the oral cavity had become healthy, we noticed the beginning of atrophy in the region 7 6 1 6 7 from the palatal side.

The first relapse began on 23 September and continued until 16 October. The temperature for this period was subfebrile at all times. Deterioration in the oral cavity set in for a few days until it appeared as though it were the indication of the beginning of exacerbation.

The second remission lasted from 16 to 30 October. The temperature was normal, further atrophy ceased, and the mucous membrane turned decidedly pale.

On 29 October, severe deterioration began in the oral cavity again accompanied by increased hyperemia in the region mentioned before and there was further baring of the roots. The second relapse began on 30 October and was more severe. It lasted until 7 December.

Only at the end of the second relapse were we able to stop further atrophy, which by that time had advanced to the point that one third of the root was bared in the region 7 6 1 6 7. The mucous membrane resumed its normal color. The marginal gingiva had drawn up, and the tongue had turned pink again. There was a daily bowel movement and the excessive perspiration ceased. The irritability also eased.

The third remission lasted from 7 December 1943 to 11 January 1944. A control examination of the oral cavity on 10 January did not show any visible changes from the end of the second relapse.

Although we examined available Russian and foreign literatures on brucellosis, we found no reference to changes in the oral cavity during the disease. Apparently this can be explained by the fact that individual instances escaped the attention of stomatologists. As Ya. S. Pekker pointed out in describing gingivitis, s. parodontitis marginalis atrophicans denu-dans, the process we described is a rare disease of the gums and etiologically has not yet been completely clarified.

The first report on brucellosis was by I. Peretokin in Sovetskaya Meditsina, No 1, 1938. We cite a quotation from this report. "With regard to the oral cavity in children with brucellosis, we found spongy bleeding gums, and an inflammation of the mucous membrane of the mouth and throat. In feverish patients the tongue was covered by a thick coating and was moist." Thus, even therapists noted changes in the oral cavity of children with brucellosis.

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Further study in the therapy of brucellosis must be conducted from our point of view by therapeutists and stomatologists together to achieve success.

On the basis of our investigations, we emphasize the importance of early sanatory measures of the oral cavity be taken along with general treatment. In the majority of cases timely treatment has checked the deterioration beyond the neck third of the root. In advanced cases the root is bared cuneiformly and further control is already impossible.

Stomatological therapy for brucellosis consists of early sanatory measures of the oral cavity --- removal of the roots, filling of decayed teeth, careful scraping off of dental tartar, rinsing with hydrogen peroxide or rivanol, and daily painting of the infected sections with 5 percent methylene blue twice a day. General therapy must be administered as well. Gum massage is a good prophylactic measure in the early stages of the disease.

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